

## AMENDMENTS TO THE SPECIFICATION

Please amend paragraph [0074] of the published application as follows:

- - ~~FIGS. 9A-9D~~ **FIG. 9** demonstrates the specificity and potency of MS-PRO Fabs by Western blot with anti-P-ERK (phosphorylated/activated ERK) antibody. **FIG. 9A** shows a dose response of MSPRO 29, 59 and 54 on RCJ-M14 cells. **FIG. 9B** shows a dose response of MSPRO 29, 59 and 54 on RCJ-W11 cells. **FIG. 9C** shows a dose response of MSPRO 29, 59 and 54 on RCJ-R1-1 cells. **FIG. 9D** shows a dose response of MSPRO 29, 59 and 54 on RCJ-R2-2 cells. - -

Please amend paragraph [0081] of the published application as follows:

- - ~~FIGS. 16A-16D~~ **FIG. 16** shows the selective binding of radiolabeled MSPRO29 to histological sections of growth plate. **FIG. 16A** shows Hematoxylin-eosin staining of growth plate treated with radiolabeled MSPRO29 at x100 magnification. **FIG. 16B** shows radiomicroscopic sections of growth plate treated with radiolabeled MSPRO29 at x100 magnification. **FIG. 16C** shows radiomicroscopic sections of growth plate treated with radiolabeled MSPRO29 at x400 magnification. **FIG. 16D** shows Hematoxylin-eosin staining of growth plate treated with radiolabeled Ly6.3 at x100 magnification. **FIG. 16E** shows radiomicroscopic sections of growth plate treated with radiolabeled Ly6.3 at x100 magnification. **FIG. 16F** shows radiomicroscopic sections of growth plate treated with radiolabeled Ly6.3 at x400 magnification. - -

Please amend paragraph [0180] of the published application as follows:

- - The combined treatment of one or more of the molecules of the invention with an anti-neoplastic or anti-chemotherapeutic drug such as doxorubicin, cisplatin or ~~taxol~~ TAXOL® provides a more efficient treatment for inhibiting the growth of tumor cells than the use of the molecule by itself. In one embodiment, the pharmaceutical composition comprises the antibody and carrier with an anti-chemotherapeutic drug. - -

Please amend paragraph [0218] of the published application as follows:

- - To express this FGFR3 variant, 293E cells (EBNA virus transfected 293 cells) were transfected with the aforementioned plasmid, pCEP-hFR3<sup>23-374</sup>TDhis, clones were identified and

grown. Cell supernatants analyzed by Western blot with anti-His antibody demonstrated high expression of the soluble receptor. Supernatants from large scale preparations were then subjected to batch affinity purification with Ni-NTA beads and the tagged soluble receptor was eluted by a step gradient ranging from 20 mM to 500 mM imidazol. A sample from each elute was loaded onto a 7.5% SDS-PAGE and stained with GELCODE GelCode® (Pierce). In parallel, Western blot analysis was performed and analyzed with anti-His antibodies. SDS-PAGE (**FIG. 1**) and immunoblot (not shown) analyses demonstrated peak amounts of purified extracellular FGFR3 in the 2nd (2) 50 mM imidazol fraction. About 0.5 mg of pure protein was obtained following this single step purification. In **FIG. 1**, T=total protein, D=dialysed protein, U=unbound fraction. - -

Please amend paragraph [0267] of the published application as follows:

- - BIACORE® and proliferation analyses showed that among the new Fabs, MSPRO54 is highly cross reactive with FGFR1. To further test the cross reactivity of the new Fabs, RCJ cells expressing either FGFR3ach (RCJ-M14; M14 on FIG. 9A) FGFR3 wild type (W 11 on FIG. 9B), FGFR1 (R1-1 on FIG. 9C) or FGFR2 (R2-2 on FIG. 9D) were incubated with increasing amount of a control antibody LY6.3, MSPRO29, 54 and 59 for one hour. FGF9 was added for 5 minutes and cell lysates were analyzed by Western blot for ERK activation (phosphorylated ERK; pERK) (~~**FIGS. 9A-9D**~~ **FIGS. 9A, 9B, 9C and 9D**). Furthermore, MSPRO13 was able to block FGFR1 activation while none of the Fabs blocked FGFR2 activation. ~~**FIGS. 9A-9D**~~ **FIGS. 9A, 9B, 9C and 9D** show the results of several Fabs, at different mg concentrations, on RCJ expressing wildtype FGFR3 or the different FGFR types. - -

Please amend paragraph [0280] of the published application as follows:

- - Femora prepared from newborn mice were incubated with 2 µg <sup>125</sup>I-MSPRO29 (17 µCi/µg) or <sup>125</sup>I-Ly6.3 (20 µCi/µg) for 1, 3 or 5 days in culture. Then, sections were processed for radiomicroscopy. After 3 days in culture, MSPRO29 was predominantly visualized at the higher hypertrophic zone and to a lesser extent at the secondary ossification region (~~**FIGS. 16A-16F**~~ **FIGS. 16A, 16B, 16C, 16D, 16E and 16F**). Hematoxylin-eosin staining of growth plate treated with radiolabeled MSPRO29 or Ly6.3 (**FIGS. 16A and 16D**, respectively) x100 magnification. Radiomicroscopic sections of growth plate treated with radiolabeled MSPRO29 or Ly6.3 (**FIGS.**

**16B** and **16E**) at X100 magnification. **FIGS. 16C** and **16F** are the same as **FIGS. 16B** and **16E** but at x400 magnification. The arrows in **FIGS. 16B** and **16C** indicate the location of the specific binding of the radiolabelled MSPRO29 to the higher hypertrophic zone of the growth plate. - -